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## Performance of Hyperkinetic and Normal Children Under Two Conditions of Reinforcement

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PERFORMANCE OF HYPERKINETIC  
AND NORMAL CHILDREN UNDER  
TWO CONDITIONS OF REINFORCEMENT

BY

HOWARD ISRAEL BENESCH

A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT  
OF THE REQUIREMENTS FOR THE DEGREE OF  
DOCTOR OF PHILOSOPHY  
IN  
PSYCHOLOGY

UNIVERSITY OF RHODE ISLAND

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## THESIS ABSTRACT

Sixteen hyperkinetics on medication (H:D), 18 on no medication (H:ND), and 20 normal control Ss (NC) (N=54) were randomly distributed between verbal and material reward groups in a 2 X 3 factorial design.

One purpose of this study was to examine one aspect of Wender's (1971) hypothesis that hyperkinetics as a group are less responsive to positive reinforcement than normal controls and that as a group, hyperkinetics on drugs should be more sensitive to reward than those off drugs. This study demonstrated that group H:D learned the task, "Golf-Ball-in-the-Hole" more efficiently than group H:ND; i.e., their performance was more similar to group NC.

Another purpose of this study was to examine the relative efficiency of verbal as contrasted with material reward with hyperkinetics, and it was predicted that hyperkinetics off drugs (H:ND) would perform more poorly under both reward conditions than groups NC and H:D. The results were suggestive: groups H:D and NC appeared to perform better on material as opposed to verbal reward; but the interaction was not significant, a finding that might have been due to excessive variability in the verbally rewarded H:ND group. However, when this latter group was discarded and groups NC and H:D were collapsed and their combined scores for verbally and materially reinforced groups subjected to a t test (one-tailed), those Ss rewarded materially performed better.

The results were discussed in terms of how they compared with previous findings, in terms of implications for future research and in terms of practical applications.

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# TABLE OF CONTENTS

Section	Page
1. Thesis Abstract.....	ii
2. Acknowledgements.....	iii
3. Table of Contents.....	v
4. List of Tables.....	vi
5. Introduction.....	1
Early Research.....	2
Laufer & Denhoff: The Second Epoch.....	8
Symptoms & Labels.....	11
Recent Objective Studies.....	35
Wender's Reinforcement Hypothesis.....	37
6. Method.....	47
7. Results.....	54
8. Discussion.....	69
9. Appendix.....	75
10. References.....	77

# LIST OF TABLES

Table	Page
1. A Comparison of Laufer and Denhoff's (1957 a, b) Original Definition of the Hyperkinetic Impulse Disorder Syndrome and Subsequent Noteworthy Studies.....	12
2. Other Symptoms Comprising the Hyperkinetic Impulse Disorder Syndrome.....	30
3. Mean Ages and Standard Deviations of the Hyperkinetic and Normal Control Groups.....	48
4. Factorial Composition of the Experiment.....	50
5. Mean Block Number in Which .80 Level of Performance Reached.....	55
6. Chi Square Analysis of Number of Subjects Requiring More Than 200 Trials to Reach Criterion of .80 Level of Performance.....	56
7. Analysis of Variance: Mean Block to Reach .80 Level of Performance.....	58
8. Mean Number of Correct Responses Per Block of 20 Trials Averaged Over 200 Trials.....	59
9. Analysis of Variance: Mean Number of Correct Responses Per Block of 20 Trials Averaged Over 200 Trials.....	60
10. Perseverator's ANOVA: Mean Number of Trials to a Correct Response After Shift of Correct Hole Had Occurred (Block #2 On).....	62
11. Analysis of Variance: Number of Trials to Correct Response After Shift of Correct Hole Had Occurred (Block #2 On).....	63
12. ANOVA: PERSEVERATORS: Mean Number of Trials to Correct Response After Shift of Correct Hole Had Occurred (Block #2 On) "Outlier Excluded".....	64
13. PERSEVERATOR'S ANOVA: Mean Number of Trials to a Correct Response After a Shift of Correct Hole Had Occurred (Block #2 On) "Outlier Excluded".....	65
14. Unweighted Means ANOVA of Correct Responses in Pairs of R-L Blocks.....	67
15. R-L Sequence - Pairs Means (Number of Correct Responses), Standard Deviations and Variances.....	68

PERFORMANCE OF HYPERKINETIC AND NORMAL CHILDREN  
UNDER TWO CONDITIONS OF REINFORCEMENT

The child diagnosed as having a hyperkinetic impulse disorder is becoming increasingly recognizable to educators, to psychologists and psychiatrists, and to other medical personnel working with children. This interest is reflected in an increasing number of studies conducted since the syndrome first began to become differentiated as a diagnostic entity approximately thirty-five years ago. Yet, today there still remains some confusion as to the diagnosis, prognosis and etiology of this syndrome. One purpose of this study, therefore, will be to review the literature in this area in order to present an integrative, precise definition of this syndrome.

The history of research on what has come to be known as the hyperkinetic impulse disorder, coined by Laufer and Denhoff (1957 a, b) is overladen with terms some of which are used synonymously with the above term. For example, Clements (1966) has identified a total of 38 terms used to describe "...a large group of children whose neurologic impairment is 'minimal' (as on a continuum), subtly affecting learning and behavior, without evident lowering of general



intellectual capacity." He introduced the term minimum brain dysfunction to describe children whose symptomatology was manifested in one or more "specific areas of brain function," without reducing general cognitive functioning to the subnormal ranges. By "minimal," Clements meant "mild," "borderline," or "subclinical." These individuals were carefully distinguished from the "culturally disadvantaged" who were seen as an "equally complex" yet "different problem."

Research in this area can be roughly divided into two epochs: one prior to the definitive work of Laufer and Denhoff (1957) when this syndrome lacked differentiation in terms of specific, observable behaviors and the other, later, when researchers began to carefully describe the symptoms comprising this complex syndrome.

#### EARLY RESEARCH

Initial studies were conducted by: Molitch and Sullivan (1937); Molitch & Eccles (1937); Bradley (1937); Bradley & Bowen (1940); Bradley & Green (1940); and Bradley and Bowen (1941). These investigators apparently were more interested in establishing the effectiveness of amphetamines with a heterogeneous group of behavior-problem children than in precisely identifying a behavioral syndrome. For example, samples were identified as "juvenile delinquents" (ages 10-17 and of "varying mental levels") (Molitch & Sullivan, 1937); "inmates of the New Jersey Home for Boys...committed by the court because of juvenile delinquency" (Molitch & Eccles, 1937 p. 587); "certain behavior problem children" (Bradley, 1937); or children "referred for a variety of neuropsychiatric complaints" (Bradley & Bowen, 1940; Bradley & Green, 1940). In addition to failing to identify a homogeneous sample,

these earliest studies suffered from other shortcomings in research strategy: lack of control groups; failure to stabilize Ss with respect to drug dosage; uncontrolled Hawthorne effects; failure to control for possible ceiling and floor effects; treatment effects subjectively evaluated, and data not subjected to statistical analysis. In sum, the efforts of earliest investigators suggested that amphetamines appeared to be helpful for a wide variety of behavior in children but failed to specify those children.

One step in that direction occurred in a work by Strauss & Lehtinen (1947) devoted primarily to the identification and education of brain-injured children. These authors introduced the concept of "minimal brain lesions" resulting from "slight noxious accidents at birth or shortly after." These lesions may be clinically identifiable shortly after their incurrence but can later be detected only on the basis of extensive psychological and behavioral observations.

The following criteria for the diagnosis of minor brain injury were presented:

1. History: evidence of pre-; peri-; or post-natal injury to the brain (inflammation or trauma).
2. Equivocal neurological signs: their presence an indication of a lesion in the brain.
3. Intellectual retardation: if present, evidence must indicate independence of that retardation from genetic factors (e.g., child from normal family, only sibling affected).
4. Perceptual and/or cognitive deficits: in absence of mental retardation.

These authors then went on to make two important observations. The first stated that minor brain damage was diagnosable solely on the basis of behavior which is similar to behavior of known brain-damaged children:

Although the first three criteria (i.e., those above) may be negative, whereas the behavior of the child in question resembles that characteristic for brain injury, and even though the performances of the child on our tests are not strongly indicative of brain injury, it may be still reasonable to consider a diagnosis of brain injury (p. 112).

This observation is very similar to that made by Gesell and Amatruda (1941): "...an entirely negative birth history and an uneventful neonatal period may nevertheless demand a diagnosis of minimal injury because of persisting or gradually diminishing signs...." (p. 231).

Thus early in the differentiation of the hyperkinetic impulse disorder the precedent was set that subjective behavioral variables (e.g., impulsivity) can outweigh quantitative data (e.g., psychometric test results) in the diagnosis of minimal brain damage. This issue has remained controversial until the present.

The second important observation was that "behavior and learning ...may be affected by minimal brain injuries without apparent lowering of the intelligence level...." (p. 128). These authors pointed out that their observations apply to children five years of age and younger; the older the child at onset of trauma or inflammation, the more similar his disability to the brain-injured adult and the less likely the chance for compensation for deficits by other parts of the central nervous system.

Some evidence corroborating Strauss and Lehtinen's hypothesis about the deleterious effects of early birth trauma on later behavior was presented by Rosenfeld & Bradley (1948). These authors, on the basis of clinical impressions, hypothesized that "a fairly uniform overt behavior pattern in maladjusted children" might be the result of "asphyxiant illness" in infancy. Examples of asphyxiant illness include pertussis, pneumonia and asphyxia neonatorum which were

hypothesized to result in six behavioral characteristics:

- (a) Unpredictable variability in mood
- (b) Hypermotility
- (c) Impulsiveness
- (d) Shortness of attention span
- (e) Inability to consistently recall material previously learned
- (f) Conspicuous difficulty in arithmetic in school

To test the validity of their impressions, these authors performed a post hoc analysis of the hospital records of 673 children. Of these, 126 had a history of pertussis occurring sometime in the first three years of life; 143 had experienced pertussis after the third year of life but prior to hospital admission; and 28 had such difficulties breathing at birth that a diagnosis of asphyxia neonatorum seemed justifiable. A control group consisting of 100 children admitted for behavioral disorders but without a history of asphyxiant illness was also selected from the 673 comprising the original sample. This sample ranged in age from 3-13 years; no mentally retarded children were included. Their behavioral problems were briefly described as "sufficient in severity to warrant hospitalization but varying widely in type of presenting symptoms." Thus this sample suffers from the same non-homogeneous characteristics reported in earlier studies and the same difficulties in generalizing obtain.

The results of this study pertain only to the sample studied and cannot be generalized to other samples as no statistical analysis of these data were undertaken; and, as the authors themselves noted, the range of their sample with respect to general intelligence and psychological functioning was restricted; i.e., only children with at

least normal intelligence requiring psychiatric hospitalization were included.

Rosenfeld & Bradley (1948) reported an association between a history of anoxia and subsequent appearance of the six behavioral symptoms in their sample. On the basis of incidence of individual symptoms, these data did not seem to strongly support the authors' contention; base rates of occurrence of these individual symptoms ranged from 22% to 57% in the control group and from 43% to 89% in the experimental groups. However, when these data were examined to determine how many children in each group had five or more individual symptoms, differences between "experimental" and "control" groups for this sample were more striking. Seventy individuals (56%) in the pertussis group, 17 children (61%) in the asphyxia nenoatorum group and seven children (7%) in the control group exhibited five or more symptoms. Hence, the incidence of the syndrome in this sample appear to be higher in the groups exposed to anoxia early in life. With respect to the pertussis group, the age at which pertussis occurred appeared to be critical. Children experiencing pertussis early in childhood in this sample (prior to fifth year) appeared more likely to develop the syndrome than thos experiencing pertussis later in life. (Note: these authors used year three as their cutoff, but such a choice does not seem to be supported by their data).

Subsequent studies, for example, Rogers et. al. (1955) and Pasamanick et. al. (1956) presented additional data linking pre-, peri-, and para-natal trauma to later behavioral difficulties.

Bradley (1950) reviewed the literature to date and summarized comprehensively the comparative effects of benzedrine vs. dexedrine

in the treatment of "maladjusted" children. He did not objectively define what he meant by "maladjusted" but did mention that it was expressed "through symptoms of restlessness, noisiness, hyperactivity and distractibility"(p. 24). He also noted that some children were maladjusted because of "underactivity and withdrawal from social contacts." The former group was "subdued" by the amphetamines, the latter stimulated "to a more active and successful social participation in conventional childhood activities." (p. 25).

Bradley then presented data accumulated since 1938 on 275 children (227 boys and 48 girls) under 13 years of age and "for the most part" of at least average intelligence. All of the criticisms of Bradley's earlier work with respect to inadequate delineation of the sample under study, qualitative treatment of data, etc., that were mentioned earlier pertain here. It should also be noted that many subjects used in this study were undoubtedly used previously so that results are not obtained from a totally different sample of youngsters and are, therefore, not completely independent of previous findings reported.

Generally, Bradley reported 50-60% of his subjects subdued by amphetamines, 15-25% demonstrating no change, 20% manifesting an increase in activity and 5% showing an improvement only in school progress. When he broke his data down according to clinical diagnosis, Bradley reported the following (ranges grouped for benzedrine and dexedrine):

Behavior Disorder	% Improved	% Unimproved	% Worse
Psychogenic in Origin (N = 221)	58-70%	22-27%	8-16%
Convulsive Disorder (N = 50)	79%	10-45%	9-13%
Psychopathic Personality (N = 32)	79-84%	4-14%	7-12%
Schizoid Personality (N = 36)	50-64%	23-29%	14-21%

These data merely suggest that amphetamines are helpful for hyperactivity that is psychogenic in origin and for that found in conjunction with psychopathy; no definitive conclusions can be drawn as these data were not subjected to statistical analysis.

#### LAUFER & DENHOFF: THE SECOND EPOCH

After thirteen years of research with "maladjusted" children manifesting either hyperactive-distractible or withdrawn-schizoid behavior, researchers had not clearly differentiated a syndrome. It was not until the work of Denhoff and Laufer and their colleagues (1957a, b) that such an attempt was made. Since these studies begin the second epoch of research in the history of the hyperkinetic syndrome, they will be reported in some detail.

Laufer & Denhoff not only labeled the syndrome but also described its component symptoms:

1. Hyperactivity: The most salient symptom identified was "involuntary and constant overactivity" present sometimes from early infancy up to six years of age and which greatly surpasses the normal. Motor development may be advanced.
2. Short attention span and poor powers of concentration: Marked by continuous shift in activity and especially noted in school.

3. Behavioral variability: Wide fluctuations in behavior so that it is difficult to predict what the child will do next.
4. Impulsiveness and inability to delay gratification.
5. Irritability: Easy expression of anger due to low frustration tolerance.
6. Explosiveness: Refers to intensity of emotional expression which is "volcanic".
7. Poor school work: Characterized by visuo-spatial problems, and exacerbated by poor concentration abilities. Learning disabilities in reading, writing (crabbed, irregular) and arithmetic are common.

It is important to realize that Denhoff and Laufer described a continuum of etiological factors ranging from "purely emotional" to singularly organic. They simply noted that the former possibility "definitely exists" but did not propose any developmental progression which would result in a hyperkinetic impulse disorder of a "purely emotional" etiology.

In contrast, much more attention was paid to organic etiological factors. These investigators, on the basis of their work with the photo-Metrazol technique of Gastaut (i.e., Laufer & Denhoff, 1954; 1957a, b) believed that this syndrome resulted from "injury to or dysfunction of the diencephalon in early life"; by "early life" these authors meant any time "...before birth, during birth or in the first five years of life...." Among the various injuries to the diencephalon (and other areas of the brain) associated with the hyperkinetic impulse disorder were: factors responsible for cerebral palsy, epilepsy, birth injuries (e.g., anoxia, high forceps delivery, severe pertussis in infancy) and organic dysfunction resulting in mental retardation.

To diagnose this syndrome these authors placed heavy weight on



case history data obtained from parents. They looked for etiological factors described above and for unique manifestations of the syndrome in the particular child under observation. A neurological examination was felt to reveal no consistent abnormalities in children with only a behavioral disorder and no "neuromotor abnormality" and/or mental retardation. EEG data were inconclusive; the authors felt that there was no close correspondence between EEG abnormalities and behavioral anomalies. Many children diagnosed as hyperkinetic manifested normal EEG's and a number of children without hyperkinesis showed abnormal EEG's.

Psychological testing appeared to be given a modest weight in their identification of the hyperkinetic syndrome. These authors felt that although such testing could reveal signs associated with brain dysfunction, there was generally "a lack of consistency of such findings with the total battery." Visual-motor functioning was almost always impaired; however, signs of brain damage usually associated with cortical involvement (e.g., perseveration, concretization) did not usually occur except in cases that had midbrain damage along with concomitant cortical dysfunction. It should be noted that the psychological test battery referred to involved the Bender-Gestalt and other tests involving drawing and a Wechsler (e.g., WISC). At this point in time, there were no data in the literature involving more standardized or sophisticated neuropsychological testing.

The last method used to identify this syndrome involved a pharmacological trial with amphetamines or barbiturates. If one obtained a positive response to the former or a negative response to the latter medication, a diagnosis of hyperkinesis was indicated. The natural disinclination of a physician to subject patients to the possible

noxious behavioral sequelae following barbiturate ingestion (e.g., increased hyperactivity) has apparently made this latter technique an unpopular one.

It should be noted that Laufer & Denhoff established a trend of identifying organic dysfunction (here primarily limited to the midbrain diencephalic structures) primarily on the basis of behavioral observations. In many cases, these observations were made by parents and were subject to all of the biases inherent in that kind of data. Not all researchers agreed with this approach (e.g., Reitan 1967); however a majority of them do.

The research literature following Laufer and Denhoff's delineation of the hyperkinetic impulse disorder syndrome was characterized by a number of individual attempts at "clarification." Table 1 lists the more noteworthy of these studies and Laufer and Denhoff's seven component symptoms. Thus the reader can get a rough approximation of how closely a given author's definition approaches Denhoff and Laufer's. Blank spaces indicate that the author(s) did not mention that particular symptom.

#### SYMPTOMS AND LABELS

In an attempt to distill an updated version of the originally defined hyperkinetic impulse disorder, each category will be briefly discussed:

##### Syndrome-Label:

The research literature since 1957 indicates roughly seventeen different ways of identifying this syndrome:

Acting-Out Child ...	1
Hyperkinetic Child ...	3
Hyperactive Child ...	8
Disturbed children who also show evidence of brain damage ...	1

TABLE 1

A Comparison of Laufer and Denhoff's (1957a, b) Original Definition of the Hyperkinetic Impulse Disorder Syndrome and Subsequent Noteworthy Studies

Study	Label	Hyperactivity	Short Attention Span	Poor Powers of Concentration
Knobel, Wolman & Mason (1959)	Acting out child	(+) (almost perpetual motion during psychiatric interview")	(-) (not found in "significant proportion")	
Burks (1960)	the hyper-kinetic child	(+) (restlessness and overactivity, motor-driveness)	(+)	
Chess (1960)	the hyper-active child	(+) ("carries out activities at a higher rate of speed than the average child")		

Behavioral Variability	Impulsive: Unable to Delay	Irritability	Explosiveness	Poor School Work
(+) ("presents a variable type of relatedness but always in contact with reality")	(+) (Excess demands; poor capacity to sustain effort)	(-) "low frustration tolerance <u>not</u> found in significant proportion"		(+) "classroom disturbance"
	(+) Poor judgment and impulsive action	(+) Low frustration tolerance and irritability		(+) Poor perceptual & conceptual abilities (reflected in serious academic deficiencies)
	(+) Impulsive: lacking in judgment			(+)

Table 1 (cont.)

Study	Label	Hyperactivity	Short Attention Span	Poor Powers of Concentration
Daryn (1961)	"disturbed children who also show evidence of organic brain damage" diffuse brain damage children	(+)	(+)	(+)
Clements & Peters (1962)	Unclear. Says "this syndrome" and refers to following: brain damage behavior syndrome; hyperkinetic syndrome; organic brain syndrome; hyperkinetic impulse disorder; Strauss syndrome; postencephalitic behavior disorder & others. Later adds: following terms used interchangeably; minimal brain dysfunction; organic learning & behavior disorders; minimal brain damage; organic nervous system deviations.	(+) Easily recognized hyperkineses; in constant motion or merely restless and fidgety.	(+) Can maintain attention only when interest aroused.	
Paine (1962)	"minimal chronic brain syndrome"	(+) At home and at school	(+)	(+) Distractibility
Prechtel & Stemmer (1962)	"choreiform syndrome" "hyperkinetic children"	(+) "excessively mobile" "restless"	(+)	(+) Inability to concentrate on anything for long.

Behavioral Variability	Impulsive: Unable to Delay	Irritability	Explosiveness	Poor School Work
	(+)	(+) Low frustration tolerance and irritability		(+) Reading difficulties
(+)	(+) Cannot help fingering objects	(+) Emotionally labile "High-strung"; irritable; aggressive; easily moved to tears.		(+) <u>Specific Learning Deficits:</u> Solitary dyslexia; dyscalculia. Cannot read at age level. Mild stress leads to dyslexic errors. Poor spelling & arithmetic. Difficulty with abstractions & part-whole relationships. Poor visuo-motor coordination.
	(+)	(+) Low frustration tolerance.	(+) Tantrums; "office wreck- ers" (purpose- less & unintentional)	(+) Reading, writing poor at first, later improve. Arithmetic a per- sistent problem. Spelling depen- dent on rote memory. Poor pattern perception, spatial relationships lead to difficulty learning to read, write.
(+) Very labile mood fluc- tuating between timid- ity & outbursts of aggression.	(+) Particularly wild unrestr- ained behavior			(+) Usual reason for referral (along with poor behavior at home)

Table 1 (cont.)

Study	Label	Hyperactivity	Short Attention Span	Poor Powers of Concentration
Anderson (1963)	No specific label mentioned; implies hyperkinetic child (-)	(+) Implied not directly stated	(+)	(+) Distractibility
Werry, Weiss, & Douglas (1964)	"hyperactive child" preferred to "hyperkinetic syndrome"	(+) Chronic, sustained severe	(+) Very short	(+) Marked distractibility to extraneous stimuli
Bakwin & Bakwin (1966)	"developmental hyperactivity"	(+) Unable to sit still	(+)	(+) Distractable
Clements (1966)	"minimal brain dysfunction syndrome"	(+) hyperkinesis; hypoactivity	(+)	(+)
Eisenberg (1966) (emphasize less severely afflicted youngster who appears normal to physician but is problem in school or with siblings)	"hyperkinetic child"	(+) "motor activity in excess of range normal for age and sex. Identifiable by history & examination. (Less severely afflicted child will be OK one to one with full attention but overactive in presence of sibs or other noisy, restless children)	(+) cannot sustain attention	(+) distractibility in perceptual

Impulsive Behavioral Variability Unable to Delay		Irritability	Explosiveness	Poor School Work
		(+) Low frustration tolerance		(+) Large number with reading & math- ematical difficulties. Disturbed visuo-motor coordination & visual perceptual difficulties.
(+)	(+)	(+)	(+) Hyperexcitabil- ity. Emotional incontinence; Poor frustration tolerance.	(+) Learning difficulties; Specific cognitive defect.
		(+) Low tolerance for frustra- tion	(+) Emotional outbursts	
(+) Excessive variation in mood & responsive- ness from day to day & even hour to hour	(+) Impulsive then remorseful	(+) Low frustration tolerance	(+) Frequent rage reactions & tantrums when crossed	(+) Mild to severe learning disability in reading, spelling, arithmetic. Poor writing skills. Poor ability organize work, confusion about dir- ctions, but succeeds with verbal ta-
		(+)		



Table 1 (cont.)

Study	Label	Hyperactivity	Short Attention Span	Poor Powers of Concentration
Levy (1966)	hyperkinetic impulse disorder; hyperkinetic syndrome; post-encephalitic behavior disorder	(+) Most striking; restless	(+) distractible	(+) Especially under school conditions
Pincus & Glaser (1966)	"minimal brain damage"			
Stewart, Pitts, Craig, Dieruf (1966)	"hyperactive child syndrome"	(+) overactive; cannot sit still fidgets		
Conners (1967)	"minimal brain dysfunction"	(+)	(+)	(+)
Denhoff (1967)	hyperkinetic impulse disorder	(+) involuntary, constant; motor development might be advanced	(+) concentration shifts from one activity to another, especially at school	(+)
Menkes, Rowe & Menkes (1967)	hyperkinetic child with minimal brain dysfunction	(+) hyperkinetic hyperactive	(+)	(+) easily distractible

Behavioral Variability      Impulsive:      Unable to Delay      Irritability      Explosiveness      Poor School Work

(+) erratic behavior; unpredictable extremes of emotional response	(+) Doing things on spur of moment. Acts before thinking.	(+) Low frustration tolerance.	(+) "Catastrophic reactions to frustrating situations".	(+) Visuo-motor difficulties combined with other difficulties lead to poor work in arithmetic & reading in spite of normal IQ and aptitude.
(+) Unpredictable		(+) Fights		
	(+)		(+) Emotional lability, aggressive	(+) Difficulties in arithmetic; problems in abstraction & in ability to work with symbols. Cardinal criterion: failure to learn despite normal IQ.
(+) Unpredictable, wide fluctuations	(+) Does things on spur of moment	(+) Low frustration tolerance. Anger easily provoked	(+) Almost vol- canic anger	(+) Problems in arithmetic (poor visuo- motor perception & poor concentration) Reversals in reading & writing; poor crabbed handwriting.
	(+)	(+) Emotionally labile, easily frustrated.		(+) More so than expected from IQ. Specific learning disorder: most commonly difficulties with abstraction, class- ification, spatial relationships, pattern perception.

Table 1 (cont.)

Study	Label	Hyperactivity	Short Attention Span	Poor Powers of Concentration
Millichap & Fowler (1967)	minimal brain dysfunction; hyperkinetic syndrome	(+) abnormally high level of motor activity; hyperexcitability	(+)	
Stevens, Boydstun, Dykman, Peters, & Sinton (1967)	MBD (minimal brain dysfunction)	(+)	(+)	
Werry (1967)	"hyperactivity" hyperactive child	(+) quantitatively in excess of normal; qualitatively inappropriate to social situation		
Paine, Wherry & Quay (1968)	minimal brain dysfunction	(+)	(+)	(+)
Werry (1968a)	hyperactive child MBD (minimal brain dysfunction)	(+)		
Werry (1968b)	developmental hyperactivity; hyperkinesis	(+) "total daily motor activity...significantly greater than the norm"	(+) extremely short	(+) easily distracted

Impulsive:  
 Behavioral Variability    Unable to Delay    Irritability    Explosiveness    Poor School Work

		(+) Low frustra- tion tolerance; aggressive be- havior.		
	(+) Motor impulsiv- ity. Inability to delay gratification.		(+) Emotional lability	(+) Specific learning disabilities
				(+) Commonest problem: non-achievement despite normal IQ. Difficulty with abstraction. Dyscalculia. Perceptual difficulties.
				(+) Coexistence of learning and be- havioral difficulties. Variety of perceptual-motor & attentional defects.
	(+) Failure to think before acting.		(+) Pleasurable or fru- strating stimuli lead to excessive excitement or anger (compared to peers or younger sibs)	(+) Cognitive disorders; Visuo-motor deficits.

Table 1 (cont.)

Study	Label	Hyperactivity	Short Attention Span	Poor Powers of Concentration
Denhoff (1969)	hyperkinetic impulse disorder	(+)	(+)	(+)
Wunderlich (1969-1970)	hyperkinetic disease; hyperactive child	(+)	(+) low attention span	(+) distractible
Werry & Sprague (1970)	hyperactivity	(+) overactive	(+)	(+)
Keogh (1971)	hyperactive children; "acting out"; "aggressive" "conduct disorder"	(+) must consider qualitative and quantitative aspects	(+)	
Wender (1971)	MBD (minimal brain dysfunction)	(+) High level of motor and verbal activity; (press of speech similar to manic adult; in older child, press of thoughts) Motor <u>hypo</u> activity and listlessness (these must have other features of syndrome & (+) response to drugs)	(+) perceptual-cognitive abnormality	(+)
Bax (1972)	hyperkinetic syndrome; hyperactivity; hyperkinesia	(+) excess motor activity	(+)	

Behavioral Variability	Impulsive: Unable to Delay	Irritability	Explosiveness	Poor School Work
(+)	(+)	(+)	(+)	(+) Learning disability associated with a disturbance in perception.
		(+) Low frustration point.		
				(+) Academically retarded.
				(+) Learning problems & poor school achievement. Educational expression of learning problems unclear, needs investigation.
(+) "predictably unpredictable"	(+) Poor "impulse control"; Anti-social behavior (e.g. sexual acting out); impaired sphincter control. Poor planning & judgment. Lack of attention to detail, disorderly, unable to finish task.	(+) Low frustration tolerance. Low threshold for regression. "Angry".	(+)	(+) Underachiever: almost a hallmark of MBD syndrome. Special learning difficulties: learning how to read, arithmetic, sloppy writing, problems in comprehension.
				(+) Specific learning disabilities.

Table 1 (cont.)

Study	Label	Hyperactivity	Short Attention Span	Poor Powers of Concentration
Van Osdol & Carlson (1972)	developmental hyperactivity (preferred term): hyperactivity, hyperkinesis	(+) motor activity more than norm for children of similar sex, mental age, socio-economical and cultural background; not associated with clear evidence of major CNS disorder or childhood psychosis present since earliest years of life	(+) extremely short	(+) easily distracted by environmental stimuli others can exclude
Weithorn (1973)	hyperactive child syndrome	(+)	(+) "limited" attention span	(+) distractible

Behavioral Variability	Impulsive: Unable to Delay	Irritability	Explosiveness	Poor School Work
	(+) Failure to think before acting.			(+) Visual-motor deficits.
	(+)	(+) Low frustration tolerance	(+)	(+) Specific learning disabilities Visuo-perceptive difficulties



Syndrome-label: (Continued)

Unclear-Polymorphus (authors use more than three different terms) ... 2  
 Minimal Chronic Brain Syndrome ... 1  
 Choreiform Syndrome ... 1  
 Development hyperactivity ... 2  
 No label mentioned ... 3  
 Minimal brain dysfunction syndrome ... 5  
 Hyperkinetic impulse disorder ... 3  
 Minimal brain damage ... 1  
 Hyperkinetic child with minimal brain dysfunction ... 1  
 Hyperkinetic disease ... 1  
 Strauss Syndrome ... 1 (used synonymously with hyperkinetic impulse disorder)  
 Hyperkinesia=hyperactivity=hyperkinetic syndrome ... 1  
 Hyperactivity=hyperkinesis ... 1

Some of these labels are discrete, some very vague (e.g., some authors used more than one term). The most popular label was "hyperactive child."

Hyperactivity:

Hyperactivity had been originally defined by Laufer and Denhoff (1957) quantitatively in terms of an amount of activity greater than "normal." Some authors continued to define it in that way; others (e.g., Wender 1971) pointed out that hypoactivity might also be a factor. Still others, e.g., Werry (1967), pointed out that the motor activity is also "qualitatively inappropriate" to its "social context." One must consider not only whether excessive activity is appropriate to a given situation but also determinants in that situation. For example, a child might behave normally in a situation where he gets exclusive adult attention but his behavior might deteriorate in a group situation with his peers where attention must be shared and where the opportunity for external control of behavior is lessened.

Also, while "activity" was generally implicitly defined as gross motor activity, some authors (e.g., Wender 1971) included press of

speech and press of thoughts under this rubric.

### Short Attention Span

Of the thirty-two studies cited, only one, Knobel et. al. (1959), concluded that a short attention span was not a defining symptom of this syndrome. The majority of authors merely noted the symptom, and five failed to mention it. One group of authors, Clements & Peters (1962), noted that although span of attention was short, it could be increased and maintained in the face of aroused interest.

### Poor Powers of Concentration

A majority of the authors mentioned this symptom; about one-third did not. Qualitatively, this symptom was portrayed as a difficulty in screening out irrelevant environmental stimuli, stimuli which others could exclude. As such, this symptom could be the result of an attentional deficit as implied by Laufer and Denhoff (1957 a, b).

### Behavioral Variability

A majority of authors, about two-thirds, failed to mention this symptom at all. Of those who did mention it, the extremes of behavioral variation were noted; i.e., behavior becomes unpredictable and erratic. Wender (1971) coined the phrase "predictably unpredictable" to describe this behavior.

### Impulsive: Unable to delay Gratification

About 40% of the authors cited failed to mention something about hyperkinetic children's impulsivity. The others mentioned something about it either cognitively, as poor judgment; [Knobloch & Pasamanick (1959); Chess (1960)], conatively, as an "inability to delay gratification"; [Knobel et. al., (1959); Levy (1966)] or motorically, as "motor

-24-

impulsivity: { Stevens et. al. (1967) }.

### Irritability-Explosive

About one-third of these authors failed to mention this symptom. Knobel et. al., (1959) did not find "low frustration tolerance" in "significant proportion" in their sample. The remaining authors reported a composite picture of a youngster who is emotionally labile or "emotionally incontinent" (Werry et. al. 1964), an easy prey for any environmental frustration. Control over these angry impulses is quite tenuous, and the youngster is often reported as hyperexcitable, easily provoked or aggressive.

### Poor Schoolwork

Fewer than 20% of the authors cited failed to mention problems in school, an observation that is in agreement with Wender's (1971) contention that "... school underachievement ... is almost a hallmark of this syndrome. One-half to two-thirds of MBD's have a learning disability." (p. 16). Wender modified his contention by stating that poor schoolwork must be evaluated in light of "capacity, motivation and adequacy of teaching"; and, therefore, poor school performance cannot be considered "a specific diagnostic sign" of MBD. Prechtel and Stemmer (1962) found school underachievement as the most usual reason for referral, Connors (1967) referred to a failure to learn despite adequate general intelligence a "cardinal criterion" of MBD. Paine, et. al., (1968) noted that non-achievement despite normal IQ is the commonest problem that they encountered.

In contrast to Wender's contention, Bax (1972) maintained that children with specific learning disabilities comprise one of seven main groups of overactive children. No data were presented and Bax

noted that his groups frequently overlapped.

#### Other

The last category comprises a group of overlapping symptoms, a result of the failure of researchers to precisely describe the hyperkinetic syndrome. These can be broken down roughly into the following categories: neurological (neuropsychological deviations, speech disorders, presence of "soft" neurological signs; abnormal EEG, especially alpha, maturational lag, poor gross and fine motor coordination, clumsiness, brain damage); developmental history (poor peri- and post-natal history); social (poor peer relationships, aggressiveness, unresponsiveness to discipline, hostile, rebellious); physiological (enuresis, encopresis, slow to toilet train); psychological (learning disabilities, visuo-spatial and visuo-perceptual difficulties, underachievement, poor short and long term memory). Table 2 gives an estimate of the incidence of these sundry, overlapping symptoms.

This table demonstrates some of the inconsistencies extant among the current authorities in this field. For example, with respect to the neurological examination, definite, though "mild" neurological signs are present according to Burks (1960), Daryn (1961), Connors (1967), Menkes et. al., (1967) and Prechtel & Stemmer (1962). They may or may not be present according to Stevens et. al., (1967); they are "minimally useful" according to Werry & Sprague (1970) and "inconclusive" according to Werry (1967).

Another area of disagreement has been the utility of the EEG in establishing the presence of this syndrome. Some researchers feel that the EEG is inconsequential in establishing diagnosis, e.g., Werry et. al. (1964); Bakwin & Bakwin (1966); Levy (1966); Werry (1967); Wender (1971). Others believe it to be minimally helpful; e.g.,

TABLE 2

Other Symptoms Comprising  
the Hyperkinetic Impulse Disorder Syndrome

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Neurological

1. Developmental History

a. Inconclusive: Stewart et. al. (1966) (no evidence for specific pre- or perinatal injury as etiological factors); Stevens et. al. (1967) (neurological signs may or may not be present; Werry (1967) (inconclusive); Werry & Sprague (1970) (minimally helpful)

b. Poor pre-, para-, or post-natal history:

Burks (1960)  
Prechtel & Stemmer (1962)  
Stewart et. al. (1966)  
Paine et. al. (1968)  
Werry (1968a)

c. Maturational Lag

Daryn (1961)  
Clements & Peters (1962)  
Clements (1966)

2. Neuropsychological Deviations:

a. Poor Flicker-Fusion performance: Burks (1960)

b. Abnormal Archimedes Spiral After Effects Test performance:  
Burks (1960)

3. Presence of "Soft" Neurological Signs:

a. Their efficacy in establishing diagnoses:

1. Helpful:

Burks (1960)	Werry (1968a, b.)
Daryn (1961)	Wender (1971)
Conners (1967)	Van Osdol & Carlson (1972)
Stevens et. al. (1967)	Weithorn (1973)
Paine et. al. (1968)	

2. Not Helpful:

Levy (1966)

Table 2 (cont.)

b. Abnormal EEG:

1. Diagnostically Important:

Burks (1960)  
Clements & Peters (1962)  
Paine (1962)  
Paine et. al. (1968) (but 59% had no abnormal EEG)  
Werry (1968a, b) (slow diffuse dysrhythmias)

2. Diagnostically inconclusive, unimportant or minimally helpful:

Werry et. al. (1964)  
Bakwin & Bakwin (1966)  
Levy (1966)  
Werry (1967)  
Werry & Sprague (1970)  
Wender (1971)

c. Motor Development:

1. Poor fine and gross motor development, clumsy:

Burks (1960)  
Clements & Peters (1962)  
Paine (1962)  
Prechtel & Stemmer (1962)  
Werry et. al. (1964)  
Clements (1966)  
Conners (1967)  
Menkes et. al. (1967)  
Paine et. al. (1968)  
Werry (1968a, b)  
Wender (1971)  
Van Osdol & Carlson (1972)  
Weithorn (1973)

2. Early motor development:

Prechtel & Stemmer (1962)

4. Speech Disorders:

Clements & Peters (1962)  
Paine (1962)  
Clements (1966)  
Stewart et. al. (1966)  
Conners (1967)  
Menkes et. al. (1967)

Social

1. Poor Peer Relationships:

Knobel et. al. (1969)  
Chess (1960)  
Clements (1966)  
Werry (1968b)  
Wunderlich (1970)  
Van Osdol & Carlson (1972)

Table 2 (cont.)

2. Aggressiveness:
 

Knobel et. al. (1959)	Werry (1968b)
Chess (1960)	Wender (1971)
Levy (1966)	Van Osdol & Carlson (1972)
3. Hostile, rebellious, resents authority:
 

Knobel et. al. (1959)
Chess (1960)
Werry (1968b)
Wender (1971)
Van Osdol & Carlson (1972)
4. Unresponsive to discipline:
 

Chess (1960)
Levy (1966)
Wunderlich (1970)
Wender (1971)
5. Porous, shallow concept of self:
 

Wunderlich (1970)
Wender (1971)
6. Emotionally labile; dysphoric, anhedonic:
 

Wender (1971)
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7. Hypo or hyper-reactive:
 

Wender (1971)
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Physiological:

1. Slow to toilet train:
 

Clements (1966)
Wender (1971)
2. Enuresis, encopresis:
 

Clements (1966)
Wender (1971)
3. Sleep disturbances:
 

Chess (1960)
Werry et. al. (1964)
Clements (1966)
Stewart et. al. (1966)
Wender (1971)

Psychological:

1. Learning Disabilities: Reading, Writing, Arithmetic, Spelling:
 

Daryn (1961)	Menkes et. al. (1967)
Clements & Peters (1962)	Stevens et. al. (1967)
Prechtel & Stemmer (1962)	Paine et. al. (1968)
Anderson (1963)	Werry (1968a)
Werry et. al. (1964)	Denhoff (1969)
Clements (1966)	Keogh (1971)
Levy (1966)	Wender (1971)
Conners (1967)	Bax (1972)
Denhoff (1967)	Weithorn (1973)

Table 2 (cont.)

2. Visual perceptive, visuo-spatial difficulties:
 

Knobel et. al. (1959)	Menkes et. al. (1967)
Clements & Peters (1962)	Menkes (1967)
Paine (1962)	Paine et. al. (1968)
Anderson (1963)	Werry (1968a, b)
Clements (1966)	Denhoff (1969)
Levy (1966)	Keogh (1971)
Conners (1967)	Wender (1971)
Denhoff (1967)	Welthorn (1973)
  
3. Underachievement:
  - Knobel et. al. (1959)
  - Levy (1966)
  - Conners (1967)
  - Werry (1968b)
  - Keogh (1971)
  - Wender (1971)
  
4. Poor short and long term memory:
  - Burks (1960)
  - Chess (1960)
  - Clements (1966)
  - Conners (1967)



Werry (1970); still others reported abnormal or borderline-abnormal records e.g., Knobloch & Pasamanick (1959); Clements & Peters (1962); Paine (1962); Conners (1967); Paine et. al. (1968); Werry (1968a, b); Van Osdol & Carlson (1972).

The presence of gross and/or fine motor impairment together with clumsiness is another symptom which is mentioned relatively often cf., Burks (1960); Clements & Peters (1962); Paine (1962); Frechtel & Stemmer (1962); Werry et. al. (1964); Clemenus (1966); Conners (1967); Menkes et. al. (1967); Paine et. al. (1968); Werry (1968a, b); Wender (1971); Van Osdol & Carlson (1972); Weithorn (1973).

Social relationships are also affected; many authors report poor peer relationships, aggressivity, and unresponsiveness to discipline (e.g., Knobel et. al. (1959); Chess (1960); Clements (1966); Levy (1966); Werry (1968b); Wunderlich (1970); Wender (1971); Van Osdol & Carlson (1972).

Learning disabilities in either visuo-spatial or visuo-perceptual areas resulting in difficulties learning how to read, to write, to spell or to do arithmetic are also frequently reported (e.g., Knobel et. al. (1959); Daryn (1961); Clements & Peters (1962); Paine (1962); Frechtel & Stemmer (1962); Anderson (1963); Werry et. al. (1964); Clements (1966); Levy (1966); Conners (1967); Denhoff (1967); Menkes et. al. (1967); Menkes (1967); Stevens et. al. (1967); Paine et. al. (1968); Werry (1968a, b); Denhoff (1969); Keogh (1971); Wender (1971); Bax (1972); Weithorn (1973). As previously mentioned, these learning disabilities occur in the presence of at least average intelligence and usually result in underachievement.

## RECENT OBJECTIVE STUDIES

There is an increasing trend toward systematically studying hyperkinetic children in the laboratory. Some of these studies provide additional information about the symptoms proposed by Laufer and Denhoff; others go beyond this definition and provide new information about hyperkinetics.

Palkes et. al. (1968), an example of the latter type of study, showed that the impulsivity demonstrated by hyperkinetic children was amenable to control by self-directed verbal command: hyperkinetic children were able to significantly improve Porteus Maze performance after they were taught to verbalize a set of self-directed commands before responding to any part of this task.

Cambell et. al. (1971) compared hyperkinetics to normal controls on four "dimensions" of cognitive style and demonstrated that they were more impulsive and more distractible than normal controls, empirical evidence for Denhoff and Laufer's definition of hyperkinetics. These authors went beyond this definition by showing that hyperkinetics were more field dependent and slower in automatization. However, this last cognitive style was not ipsatively defined as prescribed by Broverman (1960a, b) and Broverman et. al. (1966); therefore, no definite conclusion about whether or not hyperkinetic children are weak automatizers can be drawn.

Some studies go beyond Laufer and Denhoff's definition and provide entirely new information about hyperkinetics; e.g., Burks (1960); Satterfield & Dawson (1971); and Berman and McKinney (1973).

Burks (1960) reported results of studies on the Flicker Fusion Test and the Archimedes Spiral After-Effects Test. The former test uses a light which can be made to flicker at increasingly high frequencies until

the observer reports that it has "fused" into a single steady light. Normals usually report fusion at higher frequencies than those with brain damage. Burks found that hyperkinetic children reported fusion at "unusually low frequencies" (p. 20). Unfortunately, no data were presented.

The Archimedes Spiral After-Effects Test consists of a flat black disc on which is painted a white spiral. On one side the spiral goes from the center to the edge in a clockwise direction; on the other side it goes in a counter-clockwise direction. Normals report a visual after-effect of expansion and contraction (depending on the side used) after the disc has revolved several times on a turntable. Burks reported that at the fourth grade level 19 of 35 (54%) of behavior-problem children saw no illusion; 64 of 274 (24%) of normals could not see it. These groups differed significantly (statistical test used and level of significance not reported). Burks rightfully cautions against use of the above devices in predicting individual performance.

Satterfield & Dawson (1971) showed that hyperkinetic children had lower basal skin conductance, fewer and smaller nonspecific GSR's and smaller specific GSR's than those demonstrated by normal children. He interpreted his findings as reflecting a lowered excitability of the midbrain RAS and suggested that hyperkinetic behavior might be an attempt to increase exteroceptive and proprioceptive sensory input.

Berman and McKinney (1973) intercorrelated 11 WISC subtest scaled scores for 70 hyperkinetic children referred for evaluation because of teacher reports of learning inefficiencies. The product-moment correlation coefficients were subjected to factor analysis by the principal components method with varimax rotation. They reported a factor structure which differed from that reported in previous factor-analytic

studies of Wechsler scales, e.g., those by Cohen (1957, 1959), Davis (1956), and Saunders (1959), in two significant respects. First, the general factor usually found in the latter factor-analytic studies of Wechsler scales was not obtained. Second, these authors reported a fourth factor, Psychomotor Speed, which accounted for approximately 9% of the total variance in subtest scaled scores and was defined solely by the Coding subtest. Since performance on this subtest is influenced by perceptual speed, short-term memory and motor abilities--any or all of which may be impaired in hyperkinetic children--these authors felt that this subtest could provide diagnostic and remedial information. It should be noted, however, that these authors chose to retain eigenvalues less than unity in the diagonal so that the reliability of this factor is yet to be demonstrated.

The research reported to date has focused mainly on a "clarification" of the behavioral aspects of the hyperkinetic impulse disorder as originally delineated by Laufer and Denhoff (1957a, b) or has expanded this definition by showing that these children differ systematically from normals on other variables such as cognitive styles, performance on standard psychological tests, etc.

#### Wender's Reinforcement Hypothesis

Wender (1971), in contrast to the above studies, has attempted to define another main symptom of hyperkinesis. He has hypothesized that hyperkinetics possess a "decreased sensitivity to positive and negative reinforcement" (p. 195) as a result of an innate biochemical abnormality in monoamine metabolism leading to a disorder of activation. This contention, if demonstrably correct, would serve as another main symptom in the hyperkinetic syndrome described by Laufer and Denhoff (1957a, b).

To date, there is little in the literature to support or refute Wender's hypothesis. One study, Stevens et. al. (1970) did provide some support that hyperkinetic youngsters are less sensitive to positive reinforcement. These investigators studied rapid tapping in 36 hyperkinetic and 36 normal-control males (ages: 3-11; IQ's not stated). These subjects were randomly divided into three groups: the free respondents (US); those encouraged to tap rapidly (S); and those rewarded (with pennies) for increasing their rate of tapping (R). These investigators reported that control R and S groups tapped more rapidly than the hyperkinetic R and S groups ( $p < .05$ ) although the US controls tapped much more slowly than US hyperactives ( $p < .01$ ). While neither US control group increased its rate of tapping across trials, both R and S groups demonstrated significant improvement ( $p < .01$ ). Moreover, control R and S groups tapped more rapidly than the control US group ( $p < .01$ ); in contrast, no significant differences were found among the three hyperkinetic groups. These data suggest that hyperkinetics responded with "basically one response tempo, moderately fast," (p. 58) while the controls responded more appropriately. In other words, it seems that controls were more sensitive to reinforcement than hyperkinetics.

Because Wender's hypothesis has not yet been directly tested with hyperkinetics, data on other samples of hyperactive youngsters is presented. Such data, of course, provides tentative support at best as the children involved were not hyperkinetic. For example, Levin & Simmons (1962a) investigated how "emotionally disturbed children" in a residential treatment center responded to praise in a free operant situation (a marble-dropping task). They found that praise in the form of comments like "good," "fine," etc. did not serve as a reinforcer for these Ss (15 boys, age 7.2 - 11.9 years) in that they attended to the

marble dropping task for only 6-18 minutes. However, these investigators did not include a baseline period in their experimental design; i.e., 6-18 minutes might be a "long time" for these Ss.

In a follow-up study, Levin & Simmons (1962b) tried to determine whether praise was a neutral or aversive stimulus for seven of the same 15 boys in their first experiment. (These seven were members of "Group T" and were labeled in this manner because their cumulative response records in the original experiment resembled extinction curves in that these children terminated responding soon after the end of the baseline period and the onset of the period in which praise was contingent upon operant response.) They also wanted to determine whether the boys' performance in the earlier study was due to: the use of an inadequate reinforcer; to the fact that they had a short attention span; to a generalized, low frustration tolerance; or to specific resistance to extinction. Thus, they argued that an adequate reinforcer should sustain performance longer and thus provide greater resistance to extinction, an observation that implied reinforcement could be withdrawn and marble-dropping behavior maintained. If shortness of attention span were the explanation, these youngsters should attend to the task the same amount of time as previously or even less because it would no longer be a novel situation to them. Theories of frustration, according to these investigators, would suggest that withdrawing reinforcement for previously reinforced behavior should lead to frustration and result in the termination of responding and a concomitant emotional discharge.

In light of the above discussion, one would assume that selection of a reinforcer would be an important factor in the design of the study. However, such was not the case. These authors selected peanuts as an adequate reinforcer because "casual observation suggested that they

would function as a reinforcer for all of the boys and that they could be consumed in quantity without evidence of satiation." (p. 541).

Moreover, since these boys were living in a state hospital with a meager allowance for the daily food budget, it was felt that "...special foods, supplied outside of the dining room, were even more potent motivators for these boys than they would be for boys in general..." (p. 541).

These observations may indeed be correct; however, there is need for more than subjective "casual observations" to demonstrate reinforcer efficacy.

The seven boys were paired according to similarity of performance in the original experiment (Experiment A) and then divided into two experimental groups: Group F, which received only food as reinforcement and Group FP which received both food and praise. Levin & Simmons reported increases in both of their dependent variables, duration of attending to task and cumulative number of responses in Experiment B, their second study. However, Experiment A lasted for 30 minutes while Experiment B lasted for 45 minutes. Since neither of these measures was corrected for duration of experiment, the authors unnecessarily contaminated their dependent variables by this ceiling effect. One should also note that since the same seven SS were used in Experiments A and B, the results of the two experiments are obviously not statistically independent. (The authors do note that their "population" was inadequately defined). It is interesting to note the increases in the dependent variable scores on this marble dropping task in spite of possible satiation effects due to exposure to the same task in Experiment B.

While the size of the sample used in this study was too small to

permit statistical analysis, an inspection of these data allowed these investigators to conclude that Group F (food only) was superior in performance to Group FP (food and praise). These results suggest that these Ss were less sensitive to "praise" as a reinforcement, a finding which supports Wender's hypothesis. However, the above-mentioned problems in experimental design, the lack of an adequate baseline to assess reinforcer efficacy, and the use of Ss other than adequately defined hyperkinetics render this support tenuous.

Studies investigating the use of behavior modification techniques with hyperactive youngsters [e.g., Patterson (1964); Patterson et. al. (1965); Doubros & Daniels (1966); Quay et al. (1966); Knowles et. al. (1968); Stevens et. al. (1970)] have provided meager support for Wender's hypothesis. These studies merely indicate that the behavior of hyperactive youngsters is conditionable: they did not use a sample of clearly-defined hyperkinetic youngsters; did not try to assess reinforcement efficacy [as in an ABAB design where a period of unreinforced performance (baseline-condition A) is followed by a period of reinforced performance (treatment-condition B) and then the series is replicated (AB)]; and did not employ normal controls to assess differential sensitivity to reinforcement.

The first of these, Patterson (1964), an N=1 case-study of a nine-year-old male who appeared more brain-damaged than "minimally" brain-damaged (i.e., abnormal EEG and pneumoencephalogram; borderline IQ; poor Bender-Gestalt performance; history of convulsions; motor incoordination), used social and primary reinforcement along with labeling the inappropriate behavior (i.e., target behavior) in others. He reported significant decreases in conditioning scores when contrasted to base operants. However, the fact that the S was brain-damaged together



with the confounding of primary and social reinforcement precludes a clear-cut interpretation of these data.

Patterson followed up this study with another using two brain-injured hyperactive boys (one was a control S). Each child was observed from a booth for at least 10 minutes per day in his classroom, four days per week; and the following high-rate responses were selected for extinction: walking, talking, distracting and "wiggling." The experimental S was equipped with a remote control buzzer attached to earphones and was given an auditory stimulus (previously paired with candy and pennies) during each time interval in which one of the high rate responses did not occur. At the end of the conditioning trial, the S received whatever reinforcement he had "earned." Although the control S showed no change in frequency of occurrence of target behavior, the experimental S manifested a significant decrease in non-attending behavior. This reduction was maintained over a four week extinction period.

Doubros & Daniels (1966) studied the effects of reinforcement (candy) on the hyperactive behavior and play activity of six Down's Syndrome boys (age range: 8-13). Their findings essentially agreed with those of Patterson and his co-workers (1964, 1965) in that Doubros & Daniels reported a reduction in hyperactivity during the conditioning phase of this investigation, a reduction which persisted during the extinction and follow-up phase. However, failure to extend the extinction period until spontaneous recovery of hyperactive behavior could be observed and then re-conditioned and the use of a sample of questionable hyperkinetics, attenuates the impact of these findings. A control group was also lacking, but the authors did cite practical reasons why such a group was omitted.

Knowles, et. al. (1968) describes an N=1 case study involving a

boy (age 7-8; VIQ=116); developmental history unremarkable except for increasing incidents of hyperactivity). Target behaviors to be extinguished or counter-conditioned were habitual running in the hall and letter reversals in writing. Candy was used as a reinforcer. The study was very informal and no data were recorded. Improvement was gauged very impressionistically, and basic experimental controls were lacking. Nevertheless, improvement was reported and six weeks after termination of reinforcement; no hyperactive behavior or letter reversals were reported by the school or the mother. This study suffers from many of the same errors as those mentioned above, and no definite conclusions about differential sensitivity to reinforcement on the part of hyperkinetic children are possible.

Drug studies involving the treatment of hyperkinetics and hyperactives with amphetamines or tranquilizers [e.g., Knobel (1962); Epstein et. al., (1968); Cohen et. al., (1971); Conners (1971); Denhoff et. al., (1971); Sykes et. al., (1971)] have generally indicated that medication improves performance on a variety of cognitive and perceptual-motor tasks. However, none of the above studies investigated the effects of medication on reinforced performance so that conclusions with respect to Wender's hypothesis are limited. If it could be demonstrated that medication influences hyperkinetics' sensitivity to reinforcement, such findings would, of course, suggest modifications in Wender's original hypothesis.

Two studies which did investigate the influence of medication on reinforced performance, Freibergs et. al. (1968) and Freibergs & Douglas (1969), adduced evidence that did not support Wender's contentions.

Freibergs et. al. (1968) administered chlorpromazine to 36 hyperkinetic boys (age range: 6-12; mean WISC IQ=106) who were psychiatric

outpatients at Montréal Children's Hospital. Performance on two types of concept learning problems were then evaluated after initial assessment and again while on medication. Half of the Ss were on continuous reinforcement (CR group) and half on a 50% partial reinforcement schedule (PR group). Analysis of on-drug learning scores revealed highly significant differences due to reinforcement schedule: more hyperkinetics under PR were unable to reach criterion (10 consecutive correct responses in a maximum of 300 trials) than hyperkinetics under CR ( $\chi^2 = 24.26$ ;  $df = 1$ ;  $p < .001$ ); normals had significantly fewer non-solvers of concepts. Thus it would seem that hyperkinetics on chlorpromazine are more sensitive to variations in amount of reinforcement: when frequency of reinforcement drops from 100% to 50% their performance deteriorates significantly, an observation which would seem to modify Wender's hypothesis.

Freibergs and Douglas (1969) followed up their initial work with another study involving 65 hyperkinetic Ss (age range: 6-12 years; Mean WISC IQ=104; three Ss were girls) who were receiving neither drugs nor psychotherapy at or immediately preceding time of assessment. These Ss were compared to a group of normals who had no known history of behavioral problems or emotional disturbance (age range: 6-12 years; mean WISC vocabulary score not significantly different from that of hyperkinetics; three normals were females). Concept formation tasks involving number or naturalistic problems were used. As in their earlier study, these authors found no significant differences between hyperkinetics and normals under a continuous reinforcement schedule; however, significant decrements in hyperkinetics' performance were found under partial reinforcement. These persisted in retest sessions two months later. These results obtained with an independent sample of hyperki-

netics add additional credence to those of their initial study and suggest that hyperkinetics on no medication are also sensitive to variations in amount of reinforcement. These results obviously do not support Wender.

Thus the literature to date contains only three studies which directly compared hyperkinetics and normal controls with respect to reinforced performance on an objective task. However, none of these authors examined whether or not hyperkinetics are differentially sensitive to different types of reinforcement. If it could be demonstrated that hyperkinetic youngsters are more sensitive to one class of reinforcers as opposed to another, such differential sensitivity would have implications for the diagnosis and treatment of this behavioral disorder.

#### Statement of Problem

The purpose of this study was to test directly one aspect of Wender's hypothesis--that hyperkinetics as a group are less sensitive to positive reinforcement. Examining two different classes of reinforcers, material and verbal, should have implications for the diagnosis and treatment of hyperkinesis as well as further defining Wender's postulations.

These purposes are operationalized in the following hypotheses:

1. Hyperkinetics as a group are less responsive to positive reinforcement than normal controls.
2. Because drugs are assumed to normalize brain functioning in hyperkinetics, it is predicted that hyperkinetics on medication will be more sensitive to positive reinforcement than those not on drugs.

3. In an effort to more precisely test Wender's hypothesis, it is predicted that hyperkinetics off drugs will perform more poorly than normal controls under both verbal and material reward conditions.

## METHOD

### Subjects

Fifty-four subjects were used. Twenty were normal controls, eight from Peck and twelve from Sowams Elementary Schools in Barrington, Rhode Island. These Ss were selected on the basis of the following criteria:

1. No evidence of hyperkinesis; no known history of behavioral problems or emotional disturbance.
2. No evidence of any learning disability (no contact with special education services).
3. No evidence of mental retardation (based on teacher observations).

The normal controls ranged in age from 7.08 to 11.92 years. Table 3 shows the mean ages and standard deviations for the six groups; an ANOVA of these means revealed no significant differences among them ( $F_{\text{Groups}} = .9453$ ; n. s.;  $F_{\text{Reward}} = .3458$ ; n. s.; and  $F_{\text{Reward} \times \text{Group}} = .9443$ ; n. s.).

The 34 hyperkinetic children were selected from the outpatient population of the Governor Medical Center and the Governor Center School, both of which are in Providence, Rhode Island. Criteria for their

TABLE 3

Mean Ages and Standard Deviations  
of the Hyperkinetic and Normal Control Groups

Normal Controls (NC)	Hyperkinetics: On Drugs (H-OD)	Hyperkinetics: No Drugs (H-ND)
Verbal		
$\bar{X}$ Age = 9.53	8.98	9.81
S Age = 1.54	1.82	1.66
Material		
$\bar{X}$ Age = 9.44	8.71	9.34
S Age = 1.67	1.50	1.54

selection was a diagnosis of hyperkinetic impulse disorder by Eric Denhoff, M.D., together with the following: no evidence of mental retardation, no evidence of psychosis and at least three of the following six characteristics: impulsivity, aggressivity, short attention span, emotional unpredictability and presence of a learning disability. Hyperkinetic youngsters were selected from the Governor Medical Center population according to a "file sampling" procedure. Since the cases were filed alphabetically, the sampling unit selected was the filing drawer. Beginning with the first drawer and proceeding backward through every other drawer, every case within a selected drawer was screened for the above criteria.

At the Governor Center School, all cases were screened and 14 Ss selected. Dr. Denhoff is the director of this school and has personally diagnosed each of these children.

These hyperkinetic children were then divided into those currently taking medication and those not on any medication. Mothers of children in the outpatient sample of hyperkinetics were interviewed carefully after their child had participated in the experiment to determine whether and which drugs had been administered in accordance with their physician's recommendations. Teachers and the principal at the Governor Center School, who administered the medication in some cases, were queried about the current drug(s) taken, if any, by the Ss selected for participation. Drugs commonly used to control hyperkinesis were employed (e.g., benzedrine, ritalin, dexedrine).

Within the normal control and two groups of hyperkinetic children, Ss were assigned to either material or verbal reward groups. This 2 X 3 factorial design is shown in Table 4. It should be noted that a factorial design usually has equal numbers of Ss in each cell, and that



TABLE 4

## Factorial Composition of Experiment

	Groups		
	Normal Control (NC)	Hyperkinetics: On Drugs (H-OD)	Hyperkinetics: No Drug (H-ND)
Verbal			
<u>n</u> = 10		<u>n</u> = 8	<u>n</u> = 9
Type			
of			
Reward			
Material			
<u>n</u> = 10		<u>n</u> = 8	<u>n</u> = 9
			<u>N</u> = 54

the design used is unbalanced with respect to columns because insufficient Ss were available to fill all of the cells. The unbalanced aspect of this design was taken into account when these data were subjected to statistical analysis.

### Apparatus

The apparatus used was a modified version of a game called Marble-in-the-Hole which we adapted and fondly called "Golf-Ball-in-the-Hole." Golf balls were used because hyperkinetic children sometimes have visuo-spatial and visual-perceptual disabilities, and it was felt that golf balls were easier for them to handle, thus reducing the possibility of confounding due to fine motor incoordination.

The apparatus consisted of a wooden, unpainted box 18" X 15" X 8½", with two holes, two inches in diameter and six inches apart, centered in the top. A felt-lined tray, placed inside the box, was used to catch the dropped balls. Microswitches were mounted under the holes so that any ball dropped through the holes would strike them.

Responses were recorded electrically by counters and a running record of the entire performance was preserved on a paper tape by three electrically-operated styli. The hole selected and the presence or absence of reinforcement were noted.

### Procedure

Each S was run individually and tested by the same E. Those tested in the schools were removed as unobtrusively as possible from their classroom. E introduced himself and told the subject where he was going and how long the game would take to play.

Upon entering the room where the equipment was set up, S faced the apparatus. Ss in the material reward group were then told:

Here is a container full of gold balls. Choose any ball and try to guess which is the right hole to drop it in. The pennies you may receive can be saved up. These pennies can be traded in after the game is over for prizes. Here are the prizes.

E then pointed out the prizes and their cost (airplane 75¢; sky commando 75¢; candy apollo 50¢; cup and ball game 50¢; bubble gum 10¢; souvenir golf ball 50¢). The S was then questioned on how much each prize cost. This questioning was done to strengthen memory. All prizes and a price list were visible throughout the session. Specific instructions are in Appendix A.

S was then instructed to wait until a buzzer sounded before making his choice of hole. This buzzer, which remained on until the subject selected a hole, was turned off during a three second inter-trial interval and came on again at the end of that interval when the S was to choose again.

After playing the game, the subject was thanked and asked if he felt he could keep everything about the game secret. If he could, so that no one found out anything about the game, he was told he would win another prize, obtainable when the E had finished playing this game with the others. This tactic worked well; as far as could be determined from interviews, all of the Ss kept the secret.

Instructions for verbally rewarded Ss were essentially the same except that there was no mention of rewards. Verbal rewards used were: "Good!"; "Fantastic!"; "Outta Sight!"; "Fine!"; "O.K.!"; "Super!"; "Very Good!"; "HMM!"; "Beautiful!"; "Far Out!"; "Terrific!"; "Wow!"; and "Great!".

This structured procedure was adopted to circumvent some of the criticism of the Marble-in-the-Hole game. For example, this procedure eliminated the need to assess baseline hole preference and eliminated

dependent variable measures of questionable reliability and validity. The reader is referred to reviews of this literature by Parton and Ross (1965), Stevenson & Hill (1966), and Parton and Ross (1967) for a thorough discussion of these issues.

Other reasons for adopting this task were presented by Stevenson (1965) who pointed out the following criteria for task selection when working with social reinforcement:

1. The task should not be of "high intrinsic interest" if the effects of social reinforcement are to be "maximized" (p. 98).
2. The task should not have a clear end point or a visible product which might be "intrinsically motivating." (p. 99)
3. The task should "minimize the effects of earlier learning" to minimize individual differences among children (p. 99).
4. The task should permit the adult to "arbitrarily" administer supportive comments so that the child has no clear idea what constitutes correct performance (p. 99).

E recorded total number of responses to right and left holes for each block of twenty responses and then calculated percent correct responses within that interval.

A pilot study was done to determine the suitability of this task. Originally 15 trials per block were to have been used; however, the task appeared too difficult, and the blocks were expanded to 20 trials. Changes in verbal reinforcement to include expressions like "Far Out!" and "Outta Sight!" were made because reinforcements like "Good!" and "Fine!" did not seem to influence these youngster's behavior.

## RESULTS

Table 5 shows the mean block number in which each of the six groups reached a criterion of 80% correct choices in a block of 20 trials. The .80 level of performance was selected as a criterion level because such performance (16 correct choices of a possible 20 in a given block) occurs much less than one time in a hundred by chance alone when this value is generated by the binominal expansion model. However, in order to use this model, each choice must be independent of another. Since these data did not meet this criterion and since there are no known statistical techniques to estimate the probability of occurrence of non-independent, nominal events occurring in a series, the binomial expansion model was chosen in order to estimate criterion level performance. By choosing a level of alpha smaller than .01, it was felt that performance at this level could indeed be viewed as non-chance.

It should also be noted that there was one outlier, i.e., someone whose performance differs extremely from the remainder of the group, removed from each of the cells which have starred means in Table 5. These outliers either performed at a chance level for 300 trials (n=3) or perseverated in choosing one hole for 300 trials (n=1). This performance is essentially dichotomous from the remainder of the sample.

Moreover, as Table 6 demonstrates, Ss failing to reach a criterion of an average of .80 level of performance in any of the ten blocks of trials, are not systematically distributed among the cells ( $\chi^2$  cannot be calculated; three cells have an expected frequency less than five.)

TABLE 5

Mean Block Number in Which .80 Level  
of Performance Reached

	NC	H:D	H:ND
Verbal Reward	1.67*	1.71*	2.11
Material Reward	1.20	1.14*	2.25*

\*One outlier removed from each of these cells. See text for explanation.

TABLE 5

Chi Square Analysis of Number of Subjects  
Requiring More Than 200 Trials  
To Reach Criterion of .80 Level of Performance\*

	NC	H:D	H:ND	
200 Trials	19 (18.52)**	14 (14.81)**	17 (16.67)**	50
More Than 200 Trials	1 (1.48)**	2 (1.19)**	1 (1.33)**	4
	20	16	18	54 = N

\* In any of first 10 blocks, these Ss performed below .80 level.

\*\* Expected frequencies

As such, it seemed justifiable to discard these Ss for this analysis.

With due cognizance to the above comments, Table 7 presents the results of a 2 X 3 analysis of variance of the means in Table 5. It can be seen that the normal control group (NC) and the hyperkinetic group on drugs (H:D) appear to reach the .80 level of performance more quickly than the hyperkinetic group on no medication (H:ND) ( $F_{\text{Groups}} = 3.46$ ;  $df = 2/44$ ;  $p < .05$ ). Although it appears that Group NC and Group H:D performed more efficiently with verbal as contrasted with material reward, such was not the case ( $F_{\text{Groups} \times \text{Reward}} = .53$ ;  $df = 2/44$ ;  $p > .05$ ). However, this interaction might have been masked by more variance in the H:ND group. When these Ss were eliminated and the NC and H:D groups collapsed and their combined scores for verbally and materially reinforced groups subjected to a one-tailed t test, those Ss rewarded materially performed better ( $t = 2.00$ ;  $df = 20$ ;  $p < .05$ ). Thus with respect to efficiency of learning, medication would seem to beneficially effect the performance of hyperkinetics.

Table 8 demonstrates the accuracy scores or the mean number of correct responses per block of 20 trials averaged over 200 trials for each of the six treatment conditions. As can be seen in Table 9, an analysis of variance of these accuracy scores reveals no differences among the three groups ( $F_{\text{Groups}} = .45$ ;  $df = 2/48$ ;  $p > .05$ ), and no differences with respect to type of reward ( $F_{\text{Reward}} = 1.95$ ;  $df = 1/48$ ;  $p > .05$ ). Thus the differences in learning efficiency among the groups early in performance do not appear to be sustained later in performance.

An attempt was made to examine qualitative aspects of hyperkinetic



TABLE 7

## Analysis of Variance:

Mean Block to Reach .80 Level  
of Performance

Source of Variation	df	Sum of Squares	Mean Square	F
Group (A)	2	6.35	3.18	3.46*
Reward (B)	1	1.28	1.28	1.39
(Cells)	(5)	(8.60)		
Group X Reward (A X B)	2	.97	.49	.53
Within-Cells (W)	44	40.28	.92	
Total	49	48.88		

Note - Analysis done with extreme scores removed.

\*  $p < .05$ .

TABLE 8

Mean Number of Correct Responses  
Per Block of 20 Trials  
Averaged Over 200 Trials

	NC	H:D	H:ND
Verbal Reward	16.3	15.5	15.7
Material Reward	17.2	16.8	16.3

TABLE 9

## Analysis of Variance:

Mean Number of Correct Responses per  
Block of 20 Trials Averaged Over 200 Trials

Source of Variation	df	Sum of Squares	Mean Square	F
Group (A)	2	5.74	2.87	.45
Reward (B)	1	12.42	12.42	1.95
(Cells)	(5)	(19.27)		
Group X Reward (A X B)	2	1.11	.55	.09
Within-Cells (W)	48	306.19	6.38	
Total	53	325.46		

performance. Table 10 presents the mean number of trials to achieve a correct response after a shift of correct hole had occurred for each group (Block #2 on). Table 11 presents the results of a 2 X 3 ANOVA of these means; no significant results were obtained ( $F_{\text{Groups}} = .72$ ;  $df = 2/47$ ;  $p > .05$ );  $F_{\text{Reward}} = 3.69$ ;  $df = 1/47$ ;  $p > .05$ );  $F_{\text{Group X Reward}} = .30$ ;  $df = 2/47$ ;  $p > .05$ ). Thus these data suggest that hyperkinetics did not persevere in their responses more than the normal controls. It should be noted, however, that one datum in group H:ND was replaced with the mean for the remaining cases in that cell as this datum was missing due to equipment failure. One  $df$  was then subtracted from degrees of freedom for total and one from degrees of freedom for error (this procedure is suggested by Lindquist, 1956, p. 148). As Lindquist notes, this procedure yields an approximate test of significance which is reasonably exact given sufficiently large degrees of freedom for error. The above analysis was verified by IBM computer 370, Model 155 using a general linear hypothesis model (program: BMD05V).

A closer inspection of the above data revealed one subject who perseverated in making 299 responses to the right hole. This performance would alternately be scored "correct" for 20 trials and then "incorrect" for 20 trials. No other  $S$  performed in this manner. When this "outlier" was removed error variance was reduced by a factor of three. The results of the least squares ANOVA (general linear model) performed with the outlier excluded are presented in Table 12 and the means analyzed, in Table 13. Thus with the outlier excluded, subjects receiving verbal rewards perseverated more than those receiving material rewards ( $F_{R/G} = 4.34$ ;  $df = 1/46$ ;  $p < .05$ ). However, no

TABLE 10

Perseverator's ANOVA:  
Mean Number of Trials to a Correct Response  
After Shift of Correct Hole Had Occurred  
(Block #2 On)

	NC	H:D	H:ND
Verbal	1.50	2.36	1.77
Material	.85	1.15	1.21

TABLE 11

## Analysis of Variance:

Number of Trials to Correct Response  
After Shift of Correct Hole Had Occurred  
(Block #2 On)

Source of Variation	df	Sum of Square	Mean Square	F
Group (A)	2	3.02	1.51	.72
Reward (B)	1	7.75	7.75	3.69 (n.s.)
(Cells)	(5)	(12.04)		
Group X Reward (A X B)	2	1.27	.64	.30
Within-Cells (W)	47*	98.89	2.10	
Total	52*	110.93		

\*One subject assigned cell mean. See text for explanation.

TABLE 12

## ANOVA: PERSEVERATORS:

Mean Number of Trials to Correct Response  
 After Shift of Correct Hole Had Occurred  
 (Block #2 On) "Outlier Excluded"

Source of Variation	df	Sum of Squares	Mean Square	F
G/U	2	0.95	0.48	0.728
R/G,U	1	2.85	2.85	4.343*
RG/R, G, U	2	0.65	0.33	0.499
Residual	46	30.13	0.65	
Total Corrected	51	34.59		

\* $p < .05$

TABLE 13

## PERSEVERATOR'S ANOVA:

Mean Number of Trials to a Correct Response  
After a Shift of Correct Hole Had Occurred  
(Block #2 On) "Outlier Excluded"

	NC	H:D	H:ND
Verbal	1.50	1.27	1.77
Material	.85	1.15	1.21



significant differences were observed among the three groups with respect to perseverative tendencies so that excluding the "outlier" did not change this interpretation ( $F_{G/U} = 0.728$ ;  $df = 2/46$ ;  $p > .05$ ).

To check if hole preference tendencies were interfering with effects of reinforcement, a  $2 \times 3 \times 5 \times 2$  ANOVA with repeated measures was undertaken. This analysis was of the mean number of correct responses per block of 20 trials. These blocks were analyzed in pairs (R-L #1; R-L #2; R-L #3; R-L #4; R-L #5) forming a "sequence". The "R" stands for a block of 20 trials when the right hole was "correct" and the "L" when the left hole was "correct." A Statistical Analysis System Regression Procedure (1972) was used and the following three analyses of these data were undertaken: an exact least squares means analysis with outlier, an exact least squares analysis using a general linear model without outlier, and an unweighted means ANOVA. The first two analyses were done to verify the results of the last analysis. Since all three analyses yielded essentially similar results, only results of the last analysis will be presented.

Table 14 presents the results of the unweighted means analysis. An inspection of that table reveals that only the main effect for sequence is significant, a finding which indicates that learning has occurred ( $F_{SEQ} = 21.78$ ;  $df = 4/48$ ;  $p < .05$ ). Table 15 presents the five means for the R-L pairs forming the "sequence."

TABLE 14

Unweighted Means ANOVA of Correct Responses  
In Pairs of R-L Blocks

Source	df	Sum of Squares	Mean Square	F
Group	2	6.03	3.01	0.42
Reward	1	14.32	14.32	2.00
Group X Reward	2	1.31	0.65	.09
R-L	1	12.71	12.71	3.45
Group X R-L	2	7.75	3.87	1.05
Reward X R-L	1	0.01	0.01	.004
Group X Reward X R-L	2	5.43	2.72	0.74
Sequence	4	36.68	9.17	21.78*
Group X Sequence	8	6.10	0.76	1.81
Reward X Sequence	4	1.54	0.39	0.92
Group X Reward X Sequence	8	1.74	0.22	0.52
R-L X Sequence	4	3.36	0.84	1.90
Group X R-L X Sequence	8	4.35	0.54	1.23
Reward X R-L X Sequence	4	3.21	0.80	1.82
Group X Reward X R-L X Sequence	8	3.48	0.43	0.99
Corrected Total	59	108.04	1.83	

\*  $p < .05$

TABLE 15

R-L Sequence - Pairs Means (Number of Correct Responses),  
Standard Deviations and Variances

Sequence - Pair Number	Mean	Standard Deviation	Variance
#1 R-L	15.01	3.60	12.98
#2 R-L	15.94	4.14	17.14
#3 R-L	16.53	3.59	12.92
#4 R-L	17.06	3.37	11.39
#5 R-L	17.07	2.93	8.61

## DISCUSSION

### Hypotheses

The results of this study provide some qualified support for Wender's hypothesis regarding the differential sensitivity of hyperkinetics to reinforcement. With regard to hyperkinetics as a group, the evidence adduced in this study failed to support Wender; no differences in hyperkinetic performance with respect to positive rewards were found. Moreover, most measures of the effectiveness of reinforcement, qualitative and quantitative, demonstrated no differences among these groups.

When hyperkinetics were separated according to whether or not they were taking medication, there is some support for Wender; i.e., the hyperkinetic group on drugs (H:D) appeared more sensitive to reward (performed more like the normal control group, NC) than did hyperkinetics on no medication (H:ND) (See tables 5 and 7). Thus groups NC and H:D reached a criterion of .80 level of performance significantly more quickly (i.e., learned more efficiently) than group H:ND. However, although statistical significance was achieved, groups NC and H:D reached criterion level about midway in the second block of trials. In contrast, group H:ND reached criterion level early in the third block of 20 trials. Therefore, the practical significance of these findings is unclear. Nevertheless, these results do suggest that Wender's hypothesis might hold only for hyperkinetics on no medication.

Verbal reward appears to be no more effective than material reward in controlling hyperkinetic behavior. However, as previously noted, the interaction of Groups by Reward might have been masked by more variance in the H:ND group. When these Ss were eliminated and the NC and H:D groups collapsed and their combined scores for verbally and materially rewarded groups subjected to a one-tailed t test, those Ss rewarded materially did learn more efficiently. This finding suggests that hyperkinetics on medication might perform better when material rewards are administered. More research is needed before these findings could be considered definitive.

#### Comparison of these Findings to Previous Experimentation

The results of this study differ from those of Stevens et. al. (1970) who found that hyperkinetics as a group responded with one basic, "moderately fast" (p. 58) tempo to a finger tapping task while normal controls responded more appropriately, i.e., faster when encouraged to tap rapidly and when rewarded with pennies. No change in performance was noted when normal controls were allowed to freely respond. However, there was no mention of whether or not the hyperkinetic Ss were taking medication; therefore, it is impossible to compare the results of this study with those of the present one. It would have been helpful if Stevens et. al. had included a hyperkinetic group on drugs to compare with the normal control group.

Levin & Simons (1962a, b) demonstrated that praise in the form of comments like "good," "fine," etc. did not serve as a reinforcer for the 15 emotionally disturbed boys studied. However, these Ss were not adequately defined as hyperkinetic (they were referred to only as "emotionally disturbed children" in a "residential treatment center"). In contrast, the present study suggested that verbal rewards could,

indeed, serve as reinforcement for hyperkinetics. The differences in findings could be attributed to the care taken in the present investigation to insure that the reinforcers were really rewarding to the Ss (i.e., a "cafeteria" of potential reinforcers was used), to the wider range and variety of verbal rewards employed, and to the fact that Ss in this study were defined as hyperkinetics rather than merely "emotionally disturbed."

The results of this study are similar in some ways to those of Freibergs et. al. (1968) and different in some ways from Freibergs & Douglas (1969). In the former study, it was demonstrated that more hyperkinetic boys on chlorpromazine were unable to reach criterion level performance (10 consecutive correct responses in a maximum of 300 trials) when under partial (50%) reinforcement than hyperkinetics on continuous reinforcement or normal controls. In contrast, hyperkinetics on continuous reinforcement performed similarly to normal controls (i.e., no differences among number of non-solvers of the concept). The authors did not examine their data with respect to whether or not there were any differences in learning efficiency. It would have been helpful had they compared normals and hyperkinetics on drugs with respect to the trial at which criterion level performance was reached.

Freibergs and Douglas (1969), a follow-up study, used hyperkinetics on neither drugs nor psychotherapy prior to assessment. Their results are different in some ways from those of the present study. These authors found no differences in performance between hyperkinetics on no drugs and normal controls when both groups received continuous reinforcement; however, under partial (50%) reinforcement, significant decrements in the performance of hyperkinetics were found. Although

the present study did not investigate variations in amount of reinforcement, it was demonstrated that hyperkinetics on no drugs and receiving continuous reinforcement did perform significantly more poorly when compared with hyperkinetics on drugs and normal controls. These latter results are not in agreement with those of Freibergs and Douglas (1969).

Discrepancies in the results of this study with respect to Wender's hypothesis and to other research, may be due in part to the task utilized. Pilot work suggested that the task was indeed appropriate. Moreover, it satisfied many of the criteria suggested for task selection by Stevenson (1965) when trying to establish the efficacy of social (in this study, verbal) reinforcement (i.e., the task had no clear end point or visible product (under verbal-reward conditions) which might intrinsically motivate Ss; it minimized effects of earlier learning so as to minimize individual differences among children; and it allowed the arbitrary administration of supportive comments so that the S had no clear idea about what constituted correct performance.) However, there was considerable within subject variation generated by this task. This observation suggests replication with a variety of tasks (i.e., sampling tasks as a random effect instead of a fixed effect). This procedure could provide more information about the interaction of hyperkinetic performance in the presence or absence of medication and under varying types and amounts of reinforcement. An ABAB design in which baseline and treatment-condition assessment could be evaluated serially, would allow a more conclusive demonstration of the effects of reinforcement on hyperkinetic behavior. However, there was value in the task and design used in the present study.

### Practical Applications

These findings suggest that drugs given to hyperkinetics to help them control their behavior (like ritalin, dexedrine and benzedrine) are helpful in that they might allow them to respond more like normal youngsters. They would also suggest that whether or not a youngster was on or off medication would have implications for the type of reinforcement to use.

### Implications for Future Research:

Some implications for future research have already been suggested or implied by the above discussion; i.e., future studies could focus on other parameters of reinforcement such as delay of reward. Moreover, research into the possible differential effects of punishment and negative reinforcement on the behavior of hyperkinetics as contrasted with normal controls is indicated.

Since this study controled type and amount of medication in a general manner, future studies might investigate specific medications and dosage levels and their possible differential effects on hyperkinetic performance. If the medications and dosage levels were investigated in an ABAB design, results would be convincing evidence for or against Wender's hypothesis. (It should be noted that practical considerations precluded conducting the present study in such a manner.) Moreover, such findings with amphetamines would have implications for Wender's biochemical model of MED since he suggests that drugs like the amphetamines affect those areas of the brain which mediate reinforcement and thereby allow hyperkinetics to respond more normally to reinforcement (cf. Wender, 1971, pp. 163-191). However, most of Wender's data adduced in support of his speculations come from the



animal literature so that research with humans is definitely indicated. It should be noted that the present study has presented data which suggest that drugs like the amphetamines do enable hyperkinetics to perform more like normals.

APPENDIX A

DIRECTIONS: VERBALLY

REINFORCED GROUP

HI \_\_\_\_\_. MY NAME IS MR. BENESCH AND TODAY WE ARE GOING TO A SPECIAL ROOM TO PLAY A GAME CALLED GOLF-BALL-IN-THE-HOLE. IT'S A FUN GAME AND WILL PROBABLY TAKE ABOUT  $\frac{1}{2}$  HOUR TO PLAY.

HERE IS A CONTAINER FULL OF GOLF BALLS. CHOOSE ANY BALL AND TRY TO GUESS WHICH IS THE RIGHT HOLE TO DROP IT IN. I CANNOT TELL YOU IF YOU ARE RIGHT OR WRONG. YOU MUST GUESS. DO YOU HAVE ANY QUESTIONS?

OK. WE'RE READY TO BEGIN. WAIT UNTIL YOU HEAR THE BUZZER SOUND BEFORE YOU MAKE YOUR CHOICE. REMEMBER, ANY GOLF BALL WILL DO. TRY TO GUESS THE RIGHT HOLE TO DROP IT IN. READY, BEGIN.

THANK YOU FOR PLAYING THIS GAME, \_\_\_\_\_. PLEASE DO NOT TELL ANY OF THE OTHERS ANYTHING ABOUT THE GAME THAT WE PLAYED. IF YOU CAN KEEP THE SECRET, SO THAT NO ONE FINDS OUT ANYTHING ABOUT THIS GAME, YOU WILL WIN A PRIZE. YOU CAN GET THIS PRIZE WHEN I'VE FINISHED PLAYING THIS GAME WITH THE OTHERS HERE AT SCHOOL.

APPENDIX A

DIRECTIONS: MATERIALLY

REINFORCED GROUP

HI \_\_\_\_\_. MY NAME IS MR. BENESCH AND TODAY WE ARE GOING TO A SPECIAL ROOM TO PLAY A GAME CALLED GOLF-BALL-IN-THE-HOLE. IT'S A FUN GAME AND WILL PROBABLY TAKE ABOUT  $\frac{1}{2}$  HOUR TO PLAY.

HERE IS A CONTAINER FULL OF GOLF BALLS. CHOOSE ANY BALL AND TRY TO GUESS WHICH IS THE RIGHT HOLE TO DROP IT IN. THE PENNIES YOU MAY RECEIVE CAN BE SAVED UP. THESE PENNIES CAN BE TRADED IN AFTER THE GAME IS OVER FOR PRIZES. HERE ARE THE PRIZES. (POINT OUT PRIZES INDIVIDUALLY). NOTICE EACH PRIZE "COSTS" YOU SO MANY PENNIES. (EXPLAIN HOW MUCH EACH PRIZE COSTS).

LET'S SEE HOW WELL YOU UNDERSTAND WHAT I'VE JUST TOLD YOU. HOW MUCH IS THE AIRPLANE? (REINFORCE IF CORRECT, EXPLAIN AGAIN IF INCORRECT). ETC. DO YOU HAVE ANY QUESTIONS?

OK. WE'RE READY TO BEGIN. WAIT UNTIL YOU HEAR THE BUZZER SOUND BEFORE YOU MAKE YOUR CHOICE. REMEMBER, ANY GOLF BALL WILL DO. TRY TO GUESS THE RIGHT HOLE TO DROP IT IN. READY, BEGIN.

THANK YOU FOR PLAYING THIS GAME, \_\_\_\_\_. PLEASE DO NOT TELL ANY OF THE OTHERS ANYTHING ABOUT THE GAME THAT WE PLAYED. IF YOU CAN KEEP THE SECRET, SO THAT NO ONE FINDS OUT ANYTHING ABOUT THIS GAME, YOU WILL WIN ANOTHER PRIZE. YOU CAN GET THIS PRIZE WHEN I'VE FINISHED PLAYING THIS GAME WITH THE OTHERS HERE AT SCHOOL.

-77-

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